



Chronic CAD/Stable Ischemic Heart Disease

ROLE OF A SINGLE NUCLEOTIDE POLYMORPHISM OF THE C-REACTIVE PROTEIN GENE ON THE ANGIOGRAPHIC EXTENT OF CORONARY ARTERY DISEASE

ACC Moderated Poster Contributions
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Authors: *George Hatzis, Dimitris Tousoulis, Nikolaos Papageorgiou, Antigoni Miliou, George Bouras, Costas Tsioufis, Andreas Sinetos, George Latsios, Gerasimos Siasos, Christodoulos Stefanadis, Hippokration Hospital, Athens, Greece*

Background: Evidence suggests that C-reactive protein, a well known biomarker of acute phase response, is implicated in the initiation and progression of coronary artery disease. However, it is still unclear, whether specific polymorphisms on C-reactive protein (CRP) gene are related with the underlying mechanisms of atherosclerosis. In the present study we examined the impact of 3872 A>G (rs1205) polymorphism on C-reactive protein levels as well as on the severity of coronary artery disease.

Methods: The study consisted of 311 patients with angiographically documented coronary artery disease (CAD) and 160 healthy controls. The 3872 A>G polymorphism was determined by PCR and HPY CHIV restriction enzyme, while hs-CRP levels were assessed by immunonephelometry.

Results: The genotype distribution among CAD patients was: GG 131 (42.1%), AG 124 (39.8%), AA 56 (18.2%) and GG: 77(48.1%), AG: 63 (39.3%), AA: 20 (12.6%) for the control group. Importantly, AA homozygotes had a greater extent of coronary artery disease (3 vs 1+2 vessels) compared to the G allele carriers ($X^2=16.773$, $p<0.01$). Although there was a significant difference in CRP levels (mg/dl) in the control group between AA homozygosity and the GG+GA genotypes (0.555 ± 0.486 vs 1.706 ± 0.891 , $p<0.01$), the CRP levels in the CAD group were similar between AA homozygotes and G allele carriers (2.242 ± 1.503 vs 2.375 ± 1.860 , $p=0.96$).

Conclusion: The 3872 A<G polymorphism on C-reactive protein gene is strongly related with the severity of coronary artery disease, while it does not modify serum CRP levels. Our findings suggest that this polymorphism may be significantly implicated in the pathophysiology of coronary artery disease.